PROCEEDINGS OF THE ULSTER NEURO-PSYCHIATRIC SOCIETY, 1951-52

President—Dr. R. S. Allison. **Secretary**—Dr. J. H. D. Millar.

19th October, 1951, at Claremont Street Hospital:

Dr. H. Hilton Stewart and Dr. J. H. D. Millar showed three cases who had in common one very striking feature; that was loss of posterior column sensation in the upper limbs, which was out of proportion to any loss of posterior column sensation in the lower limbs.

Dr. Stewart's patient was aged 68. Her history had started with pains in the occipital region, followed by numbness in the upper limbs, progressive over a matter of weeks. She also complained of some weakness of the legs and dysuria. The history began about four months before admission to hospital, when she was slightly disorientated. The cranial nerves were normal. The upper limbs were grossly ataxic due to the loss of posterior column sensation. Distally in the hands, there was loss of light touch and pain sensation. The lower limbs showed a mild paraparesis, and minimal sensory signs. She was grossly incontinent of urine. All investigations, including lumbar myelography, were entirely negative. Her condition was beginning to mend when she was shown to the meeting.

Dr. Millar's patient was aged 45. Her history was of similar duration and also started with pains in the occipital region and numbness in the arms and slight difficulty in walking. There were, however, no bladder symptoms. Mentally she was quite clear, and at no time was she as ill as the first patient. The main feature was the gross ataxia of the upper limbs and only slight posterior column signs in the lower limbs with minimal weakness and ataxic gait. She has improved. Dr. Millar showed the third case, which was generally agreed to be a case of disseminated sclerosis with the unusual feature of greater posterior column loss in the upper limbs.

The diagnosis of the first two cases was discussed at some length. The possibility of spinal cord compression, due to tumour or platybasia, was unlikely in view of the normal myelography. The lateral distribution of the lesions in upper cervical cord and also the age of onset was unlike disseminated sclerosis. Dr. Harriman suggested the possibility of an atypical Guillan Barré syndrome even with the normal C.S.F. findings. It was generally agreed that this was probably an atypical Guillan Barré syndrome, although by no means certain.

Dr. R. S. Allison showed a very interesting patient who at one time had shown well the symptoms of autotopognosia and anosognosia. This case history will be published elsewhere.

16th November, 1951, at Claremont Street Hospital:

Dr. Millar showed two patients suffering from Unverricht's familial myoclonic epilepsy. The first, a youth aged 19, was bedridden, dysarthric, deteriorated, incontinent and having continuous myoclonic jerks. The history started at the age of 7 with attacks in which he fell frequently, especially downstairs. The condition has progressed steadily despite treatment, and in addition he had frequent major fits. The E.E.G. showed frequent sharp waves, bilaterally synchronous, associated with the myoclonic jerks. At times epileptic complexes were seen. The myoclonic jerks were increased in frequency and violence by photic stimulation. The patient is one of eleven siblings—two brothers suffer from epilepsy. One aged 17 has had three major fits and weekly akinetic attacks, when his "legs suddenly leave him" without loss of consciousness. The other brother is a bright little boy of 10, who had a year's history of major seizures, especially when roused from sleep. Both had similar epileptic complexes in the E.E.G.'s and sharp waves associated with myoclonic jerks with photic stimulation.

The second patient, a married woman of 35, had suffered from jerking movements for seven years. There was also a history of two major fits following confinements. On examination there were frequent myoclonic movements involving the facial and articulatory musculature, giving rise to an intermittent dysarthria. There were less frequent myoclonic jerks of the upper and lower limbs. The condition responded fairly well to phenobarbitone. Her father was dead, but appears to have suffered from parkinsonism, and one paternal uncle also was dead and had suffered from parkinsonism. She had six sisters and one brother, all living. Two sisters had myoclonic epilepsy, both had been examined by Dr. Millar. E.E.G.'s of the younger two sisters show epileptic discharges, bilaterally synchronous, accompanying the clinical myoclonic jerks.

Mr. R. C. Connolly read a paper entitled "Leakage of cerebro-spinal fluid masking the presence of intra-cranial hæmatomata." Four cases were described. All of these had intra-cranial hæmatomata associated with skull fractures, involving either the air sinuses (two cases) or middle ear (two cases). The intra-cranial hæmatomata produced no disturbance of consciousness and no abnormal neurological signs, while C.S.F. leaked from the nose or ears. In the cases of frontal sinus fracture, craniotomy was performed for persistent C.S.F. rhinorrhœa, and large intra-cranial hæmatomata were discovered as chance findings. In the cases of middle ear fracture, and immediately following the spontaneous cessation of the C.S.F. otorrhœa, there was a deterioration in the state of consciousness and hemiparesis appeared. In one case the hemiparesis was ipsilateral, due to a subdural hæmatoma arising from a contra-coup cerebral laceration. All cases recovered after operation and evacuation of the hæmatomata.

It was considered that the leakage of cerebro-spinal fluid facilitated the formation of intra-cranial blood clots by lowering intra-cranial tension. While the leak persisted, the presence of the hæmatomata was concealed.

28th December, 1951, at Claremont Street Hospital:

Dr. J. R. Milliken read a paper: "Psychiatric Aspects of Sleep Disorders in Children." The majority of children examined at the Child Guidance Clinic are found to be suffering from some form of sleep disorder. Organic sleep disorders are of two kinds — one, the narcoleptic syndrome, including cataplexia, sleep paralysis, and sleep hallucinosis, and the other hypersomnia. Recent claims have been made that narcolepsy is in many cases primarily a psychological disorder, and in the experience of many psychiatrists psychotherapy has been used with advantage. So-called idiopathic narcolepsy is rarely found in children.

Sleep disturbances, in which there is no apparent disease of the central nervous system, rarely occur as a single entity but are usually a part of various forms of psychological disturbance. The first type of disturbance is insufficient and restless sleep. The latter is evidenced by jerking movements, crying out, teeth-grinding, sleep-talking and jactatio capitis nocturna. The latter is not to be confused with head-banging, which usually occurs in infants or in imbecile or idiotic children. This type of disturbance is most commonly found in restless, over-active and easily excited children. Treatment must attack factors and situations which tend to drive the patient in the direction of increased motility.

True insomnia is rarely found in children except as a result of bodily discomfort. What is generally called insomnia in children is usually an unwillingness, rather than an inability, to go to sleep, and often results from faulty training in sleep habits. Much more fundamental is the sleeplessness which results from disturbing obsessive preoccupations.

Nocturnal fear reactions occurring during sleep are indicated by nightmares and night-terrors, both of which are a drastic indication that something is wrong and that the child's personality is in need of investigation and adjustment. The main differences between nightmares and night-terrors were described, and the possibility of night-terrors being due to carbon dioxide intoxication, epilepsy, hysteria or hypoglycæmia was discussed. It was felt that there was very little evidence that any of these conditions were of primary

importance, but cases had been seen in which administration of sugar or glucose had been immediately effective. They rarely, in any case, occur after the age of puberty.

Sleep-walking is nothing more than an enacted dream, and is distinguished from night-terrors by lack of affect and the purposiveness of the child's activities. Other disturbances include prolongation of the twilight state before going to, and after waking from, sleep, with hallucinations and delusions. This condition is most evident at puberty. Excessive sleep is also often found in very dull children with a limited range of interest. Enuresis has been associated from time to time with excessive depth of sleep, and although there are enuretics who benefit from amphetamine, this is by no means invariably the case.

18th January, 1952, at Claremont Street Hospital:

Dr. R. S. Allison presented the case of a man aged 50 who had been first seen on account of discomfort over the precordium. The usual investigations had disclosed no evidence of disease, and on closer enquiry it had become apparent that the patient was convinced that his wife (from whom he had been separated and not seen for many years) was indirectly responsible for his symptoms through the hostile occult activities of her relatives. Details of their magical power in this respect were given readily by the patient, and it was shown that he was the subject of auditory hallucinations, and that far from recognising the absurdity of his claims or fighting against them he was actively pursuing different lines of enquiry through Welfare Centres, etc., to try and shake off this persecution, though he himself was convinced it was hopeless. There was no evidence of personality disintegration and very little evidence of emotional tension.

There was general agreement that the case was one of paraphrenia, but that there was no present risk of the patient becoming actively dangerous to himself or others and no indication, therefore, for such a procedure as leucotomy. Dr. Allison expressed the view that there might be a small group of such cases on the borders of paraphrenia, to which the same attributes as those attached to certain cases of hypochondriasis might be applied, i.e., morbid interest and preoccupation without much evidence of severe anxiety or tension.

Dr. Hilton Stewart showed a patient, a woman aged 52. She complained of failing vision since May, 1951; she developed a right-sided ptosis in July, 1951; she became drowsy and developed a left-sided ptosis in November, 1951. Shortly after this she retired to bed and complained of frequent headaches. On examination she was mentally deteriorated and disinterested. Neurologically the picture was striking, with bilateral marked ptosis and incomplete third nerve palsies. The pupils, however, were not dilated. There was also bilateral partial nerve deafness. The blood Wassermann was negative, but the C.S.F. showed one white cell, protein 100 mgm. per cent, globulin +, W.R. + +, paretic Lange curve. The diagnosis was discussed at some length, and the consensus of opinion was in favour of meningo-vascular syphilis.

Mr. R. J. Luke read a paper on Cybernetics—"Mind and Matter." From experience we know that man's brain can study the brain of man. We have achieved useful results from such study, and as yet, the philosophical difficulties have had but little effect on the usefulness of the results. We picture the brain of man in terms of our machines, and try to understand the working of the mind, by applying the same forms of thought as are used in analysing the functions and mechanisms of machines. Man has very little in common with the calculating machines which have so far been produced. One of the most promising trends of current ideas is to consider the brain as a machine for handling information. This study of communication in such a general sense, in both man and machine, has been given the name cybernetics.

Examples of the similarities existing between some of the more recent forms of machinery, particularly electronic devices, and the functions of the human brain were given. It may be that by studying their similarities and their differences, we will eventually be able to answer the question "Mind or Machine?"

18th February, 1952, at Claremont Street Hospital:

Dr. Russell Brain read a paper on cerebral localization. He began by emphasising that present-day ideas on this subject were in the melting-pot. He reviewed the history of cerebral localization from pre-historic trephining to recent work on the frontal lobe syndrome, with special reference to prefrontal leucotomy.

He discussed at some length the conflicting opinions concerning the origin of the pyramidal tracts, quoting Fulton and his work on areas 4-6, and Walshe, whose hypothesis is that the cortex represents movement groups rather than individual muscles. He drew attention to what Walshe called the "leading parts," with special reference to Jacksonian epilepsy. Walshe states that experimental results should only be expressed in terms of the experiment.

Dr. Brain drew attention to the interesting findings in cases where hemispherectomy has been performed. He thought that it was best to regard motor cortex and cortical spinal tract as the final common pathway of skilled movements. He added that he frequently found a transient extensor plantar response resulting from lesions distant to areas 4-6.

In discussing sensation, he drew attention to the recent work which suggested that pain may have a cortical representation. Analgesia has been shown to occur as the result of cortical lesions when these are small. He pointed out that cortical stimulation of the human cortex gave rise to only crude movements and sensations. Speech, as such, has never been produced — only vocalisation similar to the epileptic cry! Aphasia from a localization point of view may be useful clinically, especially in the purer expressive and receptive types. This does not mean it is possible to localize the function of speech. He then briefly discussed the vexatious problem of cerebral dominance. He also stated that consciousness could be disturbed by lesions of the mid-thalamus, posterior hypothalamus and brain stem, and that this disturbance of consciousness should be distinguished from that arising from cortical lesions.

Summing up, he pointed out that, although symptoms may be useful for clinical localization, it did not necessarily follow that the function which was disturbed could be localized at the site of the lesion. This he illustrated by an analogy—that if we represented the function of speech as a cup, and if the cup were broken, as in dysphasia, the pieces may represent the disturbance of speech, but it was the cup as a whole which represented the normal function of speech.

21st March, 1952, at Claremont Street Hospital:

Dr. N. P. Moore read a paper on "Indications for treatment in psychiatry." He began by emphasising that there was no scientific basis for the physical treatments in psychiatry. As regards electric convulsive therapy (E.C.T.), there was only one clear-cut indication; that was a certain type of depression which was characterised by the following features: — (a) sudden onset, (b) early morning waking, (c) worse in the morning, improving as the day progresses, (d) self-reproach and a feeling of futility, (e) suicidal preoccupation, and (f) little or no variation from day to day. These were the features which characterised the endogenous depressions, involutional melancholia and manic-depressive psychosis. A great deal of suffering could result where E.C.T. was given wrongly.

For early schizophrenia deep insulin comas were the treatment of choice—at least sixty comas were necessary—the use of E.C.T. in early schizophrenia he strongly condemned.

Modified insulin therapy was useful in chronic anxiety states where there was marked tension, in cases of addiction during the stage of withdrawal and after E.C.T. in debilitated patients. Prefrontal leucotomy was a last resort treatment, and was particularly useful in chronic tension states, but did lead to personality changes. It gave good results in chronic schizophrenia and depression if the previous personality was good. In the obsessional neuroses if the rituals are firmly established the prognosis is not so good.

Prolonged narcosis was valuable in the treatment of reactive states, but the patient must be asleep for 5-14 days and 20 out of 24 hours of the day. It was essential to produce an emotional catharsis on the withdrawal of treatment.

Abreactive techniques were useful in the chronic anxiety hysterias:—He discussed pentothal, ether and carbon dioxide treatment. The latter was useful in patients who had a sound basic personality, and 30-40 treatments were required. He had had good results in two cases out of four of torticollis, 50 per cent of stammerers, 50 per cent of cases with phobias and in adult enuretics.

18th April, 1952, at Claremont Street Hospital:

Dr. McDonald Critchley, Dean of the National Hospital, Queen Square, read a paper on "Parietal lobe symptomatology." He began by emphasising that the symptomatology which resulted from lesions of the parietal lobe were paradoxical—they could be present in profusion or absent entirely. It also required a special technique to elicit them.

1. Symptoms which could result from lesions of either hemisphere were:—Cortical sensory syndromes (stereognosis, two point discrimination). Tactile inattention. Hemianæsthesia. Pseudothalamic syndrome. Hemiatrophy (patchy wasting of arm and leg). Visual inattention. Constructional apraxia (both hands). Unilateral visual disorientation. Pseudo-cerebellar ataxia.

2. Lesions of dominant hemisphere only:—Alexia. Apraxia (bilateral). Visual autotopognosia. Gerstmann's syndrome (1924) (Difficulty writing and counting. R. & L. unawareness. Finger agnosia. Acalculia). Pain asymboly. Anæstho-agnosia. Bilateral sensory loss.

3. Non-dominant hemisphere:—Contralateral loss of body image. Anosognosia. Loss of extra-personal space on left.

4. Bilateral lesions:—Visual disorientation. Constructional apraxia. Plano-topokinesia.

The above states were explained and examples illustrating them were given.